In the Specification

Please substitute the following Title of the invention on page 1, lines 5-6:

METHOD FOR THE [MODULATION] INHIBITION OF
FUNCTION OF STAT TRANSCRIPTION FACTORS

Please substitute the following paragraph beginning on page 1, line 11:

[This work was supported in part by grants from the US Public Health Service, the National Institutes of Health and the National Cancer Institute] This invention was made with government support under National Institutes of Health grant number NCI 5 RO1 CA56072. The government has certain rights in the invention.

Please substitute the following Cross-Reference to Related Applications paragraph, on page 1, after the Title:

[This patent application is a National Phase Concerning a Filing Under 35 U.S.C. 371, claiming the benefit of priority of PCT/US99/17366, filed July 30, 1999, which claims the benefit of priority of U.S. Provisional Application Serial No. 60/094,695, all of which are incorporated herein by reference] This application is a U.S. national stage application of international patent application No. PCT/US99/17366, filed July 30, 1999, which claims the benefit of U.S. Provisional Application Serial No. 60/094,695, filed July 30, 1998.

Please substitute the following paragraph beginning at page 2, line 25, through to page 3, line 1:

A third method involves dominant-negative mutant transfections. This includes the transfection of cDNA encoding non-functional mutants of specific transcription factors or proteins that interact with the transcription factors[,]. [such] Such mutants are non-functional, and also interfere with the function of the normal endogenous transcription factor within the cells. These have the disadvantage of the technical difficulty of performing the transfections, isolating the cells that actually are expressing the dominant-negative protein, and regulating the level of expression of the dominant-negative protein in the cells.

Please substitute the following paragraph beginning at page 3, line 8:

There is provided a method of modulating the function of a transcription factor by administering an effective amount of an oligonucleotide containing optimal nucleotide binding sites for the transcription factor. A therapeutic agent having an effective amount of an oligonucleotide for modulating function of transcription factors and a pharmaceutically acceptable carrier is also provided. There is provided [~~an~~] oligonucleotides having transcription factor modulatory properties. Also provided is a treatment of patients having illnesses in which the activation of transcription factors play a role, by administering to a patient an effective amount of an oligonucleotide which competitively binds the related transcription factor. Also provided is a method of determining prognostic factors associated with particularly malignant cells by determining if particular transcription factors are constitutively activated.

Please substitute the following paragraphs beginning at page 3, line 32, through to page 5, line 24:

Figure 1 shows the sequence of the sense strand of double-stranded DNA fragments that were used for gel mobility assays and for the inhibition of the activation of STAT5;

Figure 2 shows the constitutive activation of a STAT-like DNA-binding factor in Dami/HEL and Meg-01 cells; Nuclear extracts from each cell line cultured in the absence of cytokine (CTL) or in the presence of as much as 400 ng/ml TPO or 40 ng/ml IL-3, were incubated with [^{32}P]-labeled, double-stranded IRF-1 GAS (SEQ ID NO. 2) oligonucleotide; The DNA-binding complex and unbound probes were separated electrophoretically on 5% non-denaturing polyacrylamide gels; The autoradiograph shows the STAT-like DNA-binding factor (DBF) and the nonspecific bands (NSB);

Figure 3 shows the identification of the constitutively activated DNA-binding factor in HEL/Dami and Meg-01 cells by gel electrophoretic mobility supershift assays; Nuclear extracts from HEL/Dami and Meg-01 cells without cytokine exposure were incubated with the [^{32}P]-labeled MGFe probe (SEQ ID NO. 1) plus anti-STAT3 antiserum (S3), or with the [^{32}P]-labeled probe plus anti-STAT5 antiserum (S5); The autoradiograph shows the STAT DNA-binding factor (DBF), nonspecific bands (NSB) and free IRF-1 GAS probe (P) (SEQ ID NO. 2); The arrowheads indicate the supershifted complexes, these results are representative of three separate experiments; Nuclear

extracts also were incubated with the [^{32}P]-labeled MGFe probe (SEQ ID NO. 1) plus anti-STAT1, anti-STAT2 and anti-STAT6 antiserum, no supershifted complexes were observed;

Figure 4 shows the effects of IRF-1 GAS (SEQ ID NO. 2) on the binding of STAT 5 to the [^{32}P]-labeled MGFe (SEQ ID NO. 1); Nuclear extracts from HEL/Dami and Meg-01 cells were incubated with the [^{32}P]-labeled MGFe probe (SEQ ID NO. 1) without IRF-1 GAS (NONE) (SEQ ID NO. 2) or with [^{32}P]-labeled probe plus a 100-fold excess of unlabeled oligonucleotide SIE (SEQ ID NO. 9) or IRF-1 GAS (I-GAS) (SEQ ID NO. 2), respectively; The DNA binding ~~compleses~~ complexes were separated in a 5% non-denaturing polyacrylamide gel; The autoradiograph shows the STAT transcription factor (STAT5) and nonspecific bands (NSB);

Figure 5 shows the effects of IRF-1 GAS double-stranded oligonucleotide (SEQ ID NO. 2) on HEL/Dami and Meg-01 cell survival and proliferation; HEL/Dami and Meg-01 cells were incubated with the ~~indicatied~~ indicated concentrations of IRF-1 GAS oligo (SEQ ID NO. 2) or SIE control oligo (SEQ ID NO. 9) with lipid for 72 hours and labeled with 2μ Ci/ml [^{3}H]-thymidine (TdR); TdR incorporation into newly synthesized DNA was determined by counting the radioactivity (CPM) from triplicate samples and expressed as the mean CPM; and

Figure 6 shows the effects of IRF-1 GAS double-stranded oligonucleotide (SEQ ID NO. 2) on HEL/Dami cell survival and proliferation; HEL/Dami cells were incubated with the indicated concentrations on STAT1 (SEQ ID NO. 10), STAT3 (SEQ ID NO. 11), MGFe (SEQ ID NO. 1), STAT5/6 (SEQ ID NO. 3) oligo or no oligo control (CTL) with lipid for 72 hours and labeled with 2μ Ci/ml [^{3}H]-thymidine (TdR); TdR incorporation into newly synthesized DNA was determined by counting the radioactivity per minute (CPM) from triplicate samples and expressed as the mean CPM.

Please replace original pages 1-3 (Sequence Listing) with new pages 1-3 attached hereto.